SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK

In Re: NEW YORK CITY ASBESTOS LITIGATION	Hon. Joan A. Madden, J.S.C. (Part 11)
This Document Relates To:	Index No. 114120/06
CHRISTIAN HOLINKA, Plaintiff -against- A.W. CHESTERTON COMPANY, et al., Defendants.	**AFFIRMATION OF TIMOTHY J. FRASER, ESQ. IN SUPPORT OF MOTION IN LIMINE TO PRECLUDE EVIDENCE OF EPIDEMIOLOGICAL STUDIES**

TIMOTHY J. FRASER, being of full age, affirms under penalty of perjury as follows:

1. I am an attorney at law of the State of New York and am an associate at the law firm of Drinker Biddle & Reath LLP, attorneys for defendant Baxter Healthcare Corporation. I have personal knowledge of the facts set forth herein.

2. I submit this affirmation in support of the motion *in limine* of defendants Baxter Healthcare Corporation (alleged to be a successor in interest to American Hospital Supply Corp. and American Scientific Products), ManorCare Health Services, Inc. (alleged to be a successor in interest to Central Scientific Company, a division of Cenco, Inc.), Fisher Scientific International Inc., VWR International, Inc. and Univar USA Inc. (collectively, "Defendants") to preclude plaintiff from introducing evidence of epidemiological studies regarding an increased risk of mesothelioma from asbestos unless Plaintiff establishes that he was exposed to similar or greater amounts of the same type of asbestos.

3. No previous application has been made for the same or similar relief sought herein.

4. Exhibit A is a true and complete copy of the July 18, 2007 report of Robert C. Adams, M.S., C.I.H., C.S.P., Defendants' industrial hygiene expert.

5. Exhibit B is a true and complete copy of the July 30, 2007 report of Kenneth A. Mundt, Ph.D., Defendants' epidemiology expert

6. Exhibit C is a true and complete copy of the July 24, 2007 report of Sheldon H. Rabinovitz, Ph.D., C.I.H., Defendants' industrial hygiene expert.

7. Exhibit D is a true and complete copy of the transcript from the February 22, 2007 deposition of plaintiff Christian Holinka

8. Exhibit E is a true and complete copy of the transcript from the March 1, 2007 deposition of plaintiff Christian Holinka.

9. I certify that the foregoing statements made by me are true. I am aware that if any of the foregoing statements made by me are willfully false, I am subject to punishment.

Timothy J. Fraser

Dated: August 22, 2007

Exhibit A

ENVIRON

July 18, 2007

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RE: **New York City Asbestos Litigation**
Christian Holinka
Index No. 114120-06

Dear Sirs and Madams:

Thank you for the opportunity to review this matter and conduct an industrial hygiene assessment of the potential asbestos exposures in this case. It is my understanding that the Plaintiff in this matter, Dr. Christian Holinka, claims that his mesothelioma is the result of exposure to laboratory materials that he associates with having contained asbestos and which he handled over the course of his academic and professional career. I have been retained by Defendants ManorCare Health Services, Inc. (alleged to be a successor in interest to Central Scientific Company, a division of Cenco, Inc.) ("ManorCare"), Fisher Scientific International Inc. ("Fisher"), Baxter Healthcare Corporation (alleged to be a successor in interest to American Hospital Supply Corp. and American Scientific Products) ("Baxter"), VWR International, Inc. ("VWR") and Univar USA Inc. ("Univar") (collectively, the "Lab Supply Defendants") to render opinions related to potential asbestos exposures, if any, that might have arisen during the activities that Dr. Holinka undertook related to the use of certain

laboratory materials, specifically mittens and Bunsen burner pads, and to assess the possibility that there may have been alternative exposures that might explain the development of his mesothelioma.

In the preparation of this report, I have reviewed the following documents supplied to me in this matter or in related matters that have relevance to this case:

Document Provided	Description	Date
Moline Report	Dr. Jacqueline Moline expert report	03/08/2007
Answers to Interrog	Letter of Application (complaint) and Plaintiff's Answers to Interrogatories	10/03/2006
Social Security Records	Social Security Records	Various
Holinka Depo I	Deposition under oral examination of Christian Holinka	02/12/2007
Holinka Depo II	Deposition under oral examination of Christian Holinka (Volume II)	02/22/2007
Holinka Depo III	Deposition under oral examination of Christian Holinka (Volume III)	03/01/2007
Plaintiff's Expert Report	Dr. James Strauchen, MD expert report Expert for the Plaintiff Pathologist	04/30/2007
Holinka CV	Curriculum Vitae of Christian Holinka	06/22/2006
Medical Records - Dr. Meyers	Medical Records from Dr. Robert Meyers	11/21/2006
Medical Records – NY Presbyterian Hospital	Medical Records from New York Presbyterian Hospital	
Medical Records – Dr. Taub	Medical Records from Dr. Robert Taub at the Herbert Irving Cancer Center	
Medical Records – Radiology	Medical Records from Columbia Presbyterian Eastside Radiology	02/01/2007
SSN Records	Social Security Records	
Workplace simulation report – Dr. Longo	The use of asbestos containing gloves: a work practice study (supplied in the matter of Thames v. Fisher Scientific)	08/2001

These documents provided information on the plaintiff's activities in academic, part-time employment, and full time employment settings. This report is intended to render an opinion on the sources of exposure to asbestos containing materials (ACM) and what contribution, if any, materials provided by the Lab Supply Defendants would have had on that exposure.

Qualifications

I am a nationally certified safety professional and certified industrial hygienist with more than 26 years experience as a safety and health professional. I have been involved in the assessment of asbestos exposures in numerous industries, including laboratories. I have had first hand experience with the laboratory products described in this case through my own academic training. During my time as the Director of Environmental Health and Safety for the New York City Department of Design and Construction, a public works agency

responsible for the construction and renovation of the majority of New York City's publicly owned buildings and infrastructure, I was involved in work in several city-owned laboratory facilities. I have conducted industrial hygiene studies of many different industrial workplaces, including laboratories and educational facilities. I have conducted a number of air sampling studies for the presence of asbestos in many different types of buildings. I carry United States Environmental Protection Agency (USEPA) and New York State Certificates as an Asbestos Building Inspector and Asbestos Project Designer and am intimately familiar with the uses of ACM in buildings, laboratory products and in other applications. I am a Fellow of the American Industrial Hygiene Association, a Professional Member of the American Society of Safety Engineers and a Diplomate of the American Academy of Industrial Hygiene. I am an adjunct professor at the Master's level teaching courses in industrial hygiene at St. Joseph's University in Philadelphia. My Curriculum Vitae is attached.

My opinions in this matter are stated within a reasonable degree of professional and scientific certainty.

Overview of Life and Work History

Dr. Holinka was born on July 7, 1937 in Germany and was a lifelong non-smoker. He immigrated to the United States in 1956 and worked briefly as an elevator operator before enlisting in the U.S. Army in that same year. After completing basic training, Dr. Holinka was stationed at Fort Sam where he was trained as a medical laboratory technician. From 1957 to 1959, Dr. Holinka worked in a medical laboratory while stationed at the 98 General Hospital. Dr. Holinka left military service in 1959 and worked for Booth Memorial Hospital for three to five months before enrolling as a student at the University of California at Berkeley. While an undergraduate, Dr. Holinka also worked part time in a research laboratory. Dr. Holinka then enrolled as a graduate student in biology at Hunter College, but transferred to medical school at McGill University after two semesters. In 1964, Dr. Holinka withdrew from medical school and started working full time for the same laboratory he had worked while an undergraduate. That same year, Dr. Holinka enrolled as a graduate student in physiology at UC Berkeley. After completing his Master of Science in physiology, Dr. Holinka enrolled as a graduate student in comparative literature and continued taking classes and working as a teaching assistant until 1971. In 1971, Dr. Holinka enrolled as a graduate student in biological sciences at the State University of New York at Stony Brook (SUNY Stony Brook). He was awarded his doctorate in 1974 and worked as a post-doctoral fellow from 1974 to 1977. Following his post-doctoral fellowship, Dr. Holinka worked as a research instructor and professor until 1989 at Mt. Sinai Hospital. From 1989 to 1996, Dr. Holinka was employed in the pharmaceutical industry with various companies. Since 1996, he has continued working with the pharmaceutical industry as an independent consultant.

In August of 2006, Dr. Holinka was diagnosed with malignant pleural mesothelioma.

Overview of industrial hygiene assessment of this case

In reviewing the information in this case, I have assessed the peer reviewed literature relative to the potential levels of exposure that would be associated with the use of the laboratory materials that Dr. Holinka alleges to have handled in the course of his academic studies, part time and full time employment, post graduate research, and faculty research. He claims that

his asbestos exposure occurred throughout his academic and occupational career. From his deposition testimony, I have assessed the exposure factors (time, frequency of use, duration of exposure and opportunities for exposure) that might have led to asbestos exposure. Based on this information, I calculated a reasonable maximum estimate of his potential average daily and lifetime cumulative exposures from the use of certain products that Dr. Holinka allegedly handled, including mittens and Bunsen burner pads, and that he alleges contained asbestos. I compared the reasonable maximum estimates to available data on cumulative exposure studies associated with the development of mesothelioma. I also compared this to cumulative levels that are associated with lifetime exposures to asbestos in the ambient environment as well as the level that a worker exposed at the current Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL) would have over a working lifetime.

The industrial hygiene approach

As defined by the American Industrial Hygiene Association (AIHA), industrial hygiene (IH) is "the science and art devoted to the anticipation, recognition, evaluation, and control of those environmental factors or stresses arising in or from the workplace which may cause sickness, impaired health and well being, or significant discomfort among workers or among citizens of the community" (http://www.aiha.org/Content/AboutAIHA/whatisIH.htm). The anticipation and recognition functions of industrial hygienists are supported by reviews of the relevant scientific literature and by familiarity with various workplaces and work practices. The evaluation function is supported by visual inspections of work places and practices, knowledge of the processes, the sources of emission, and by objective measurements of the agent of concern. Such objective measurements can be obtained from peer-reviewed studies of the specific tasks or studies of analogous tasks that have similar exposure conditions or through contemporary field measurements or workplace simulations. A critical aspect of the industrial hygiene approach is identifying and implementing measurement techniques and analysis methods appropriate to the type of compound of interest and considering the potential interferences that can arise from the work environment.

When properly performed, the results of the workplace measurements can be compared to regulatory standards, such as the OSHA PEL, or consensus non-regulatory guidelines, such as the American Conference of Governmental Industrial Hygienists Threshold Limit Values. However, the assessment process is not limited to the comparison of results to standards or guidelines. The appropriate interpretation of exposure measurements includes an assessment of the magnitude of human health risks experienced by individuals with exposures at various levels and for different amounts of time. This assessment includes calculating a likely range of exposures experienced by individuals under different circumstances, and comparing those exposure estimates to exposure levels that have been associated with health risks as described in the literature.

If the potential for an unacceptable level of exposure is identified in the course of an evaluation, IH practitioners also posses the training and expertise to recommend appropriate and practical methods to reduce or eliminate exposure through engineering, environmental, or administrative controls, or the use of personal protective equipment.

Exposure estimation using IH data

From the IH perspective, the assessment of an individual's asbestos exposure is evaluated based on the following factors:

- exposure magnitude (concentration in the air);
- exposure intensity (hours of exposure each day or week); and
- exposure duration (number of years of exposure).

With respect to asbestos-related diseases, it is also essential to account for fiber type and fiber size in order to characterize health risks appropriately.

Exposure magnitude, intensity and duration

Industrial hygienists typically summarize exposure with measures that integrate magnitude, intensity and duration of exposure. The magnitude of exposure to asbestos is generally measured in units of fibers per cubic centimeter of air (f/cc). When multiplied by the intensity of exposure, the result is the daily average or time-weighted average (TWA). For the assessment of occupational exposures, an 8-hour workday is assumed. Cumulative exposure metrics additionally account for the exposure duration, in years, and are typically expressed as fiber-years/cc or sometimes simply fiber-years. For example, a daily TWA exposure of 0.1 f/cc for 1 year would result in a cumulative exposure of 0.1 fiber-years/cc.

Lifetime cumulative exposure associated with employment is considered to occur over 40 to 45 years (i.e., the expected duration of a person's working life, if employment begins at age 20 and ends at age 60 or 65). The lifetime cumulative asbestos exposure of a worker employed for 40 years at the current OSHA PEL of 0.1 f/cc (OSHA 1994) will be 4 fiber-years/cc. Asbestos-related lung diseases (malignant and nonmalignant) or signs of these diseases have been reported in groups of occupationally exposed humans with cumulative exposures ranging from about 5 to 1,200 fiber-years/cc. Such cumulative exposures would result from 40 years of occupational exposure to concentrations ranging from 0.125 to 30 f/cc. (ATSDR 2001).

Small quantities of asbestos fibers are ubiquitous in air, arising from natural sources, windblown soil from hazardous waste sites, deterioration of automobile clutches and brakes, or breakdown of asbestos-containing materials such as insulation (ATSDR 2001). In some urban environments, the ambient concentration of asbestos has been reported to be as high as 0.001 f/cc (IPCS 1986). For a 70-year lifetime, this would result in a total cumulative exposure of 0.07 fiber-years/cc. Such a cumulative level of exposure is not known to be associated with any increased risk of asbestos related disease.

Fiber type

Chrysotile asbestos is unique in that it has a serpentine fiber-formation (curled fibers) compared to the amphibole fibers, including amosite and crocidolite, which are straight and needle-like. Chrysotile asbestos is less likely to be retained in the lung if inhaled with a short half-life on the order of weeks or months. In contrast, amphibole fibers have a half-life in the lung of 20 to 40 years. Amphiboles are thus considered to be more biologically active than chrysotile (Rasmuson 2004) and have been found to be more strongly and consistently

associated with risk of mesothelioma, compared with chrysotile, in persons occupationally exposed to asbestos (Hodgson, 2005).

According to a 1988 review by Churg, although chrysotile asbestos may produce mesothelioma in man, the total number of such cases is small and the required doses are extremely high. Some studies suggest that the cumulative lifetime exposure to chrysotile would need to be in the range of 25 fiber-years/cc to 100 fiber-years/cc (Rasmuson 2004). In the absence of any amphibole exposure, the risk of mesothelioma from exposure to chrysotile fibers alone, especially in low concentrations, is considered insignificant.

Chrysotile is the most common form of asbestos used in the United States, making up nearly 99% of all asbestos products that were produced (NIOSH/OSHA 1980; ATSDR 2001). Vitra (2005) reported that chrysotile accounted for 96% of the world production and consumption of asbestos products from 1900 to 2003. Laboratory products that have been studied, such as asbestos gloves, have been found to contain only chrysotile. It is likely that products like Bunsen burner pads would also be chrysotile containing since the amphiboles tend to be more inflexible and thus are more limited in being fabricated into products (ATSDR 2001). The white color of the center is also a indication that the product is chrysotile containing; chrysotile fibers are white, amosite is yellowish-brown, and crocidolite is a lavender or blue color (Vitra 2005). The amphibole forms, based on information in the literature and my personal experience in the inspection and sampling of buildings for the presence of ACM, are more commonly associated with friable insulation materials, especially steam pipes and boilers. Amosite was commonly used in marine vessels (Harries 1971) and industries with hot processes such as steel mills. Crocidolite was also used in some marine vessels (Harries 1971) and was used in gaskets associated with acid piping in pulp and paper mills (Mangold 2006).

Fiber size

The final determinant of risk for the development of asbestos related diseases is fiber size. Studies have consistently shown that long thin fibers greater than 5 microns in length with an aspect ratio (length to width ratio) equal to or greater than 3:1 present the greatest risk of mesothelioma development (ERG 2003). Particles that do not meet these size parameters are not known to be associated with an increased risk for the development of asbestos related diseases.

Exposure estimation

The first step in the estimation of the exposure that an individual may have received is a careful review of the scientific literature for information on historic exposures associated with the job or task that the person performed. A tool that is used for this purpose is PubMed, provided by the US National Library of Medicine. PubMed is a powerful standard research tool available free over the internet, which can search the scientific literature published since about 1966. Publications available via the NIOSH, OSHA and USEPA websites can also provide valuable historic exposure information. Where there is no specific data available for a particular work task, the industrial hygienist will use estimates from jobs with similar exposure conditions or work practices. In addition, there are also general groupings of exposure values associated with the use and handling of certain types of materials. For example, work involving non-friable asbestos materials have had historic airborne concentrations that range from 0.01 to 0.1 f/cc, with many data in the ambient

background range (Rasmuson 2004). If an individual is working with a non-friable material, then the industrial hygienist will assign a value based on the nature of the work that is being performed, selecting the lower end of the range for work that involves limited opportunities for disturbance and selecting the higher end of the range if the work involves tasks with more potential for disturbance. From this information, an exposure value (or range of values) is assigned to each job or task and, with the exposure time factors, is used to provide a reasonable maximum estimate of the daily 8-hour TWA. When multiplied by the duration of exposure, the individual contribution to the cumulative lifetime exposure can be determined for each task or job. The sum of the individual contributions is the total cumulative lifetime exposure for the individual, which can then be compared to information related to the lifetime risk for the development of disease.

Opportunities for Exposure

U.S. Army

Dr. Holinka trained and worked as a laboratory technician in the U.S. Army from 1956 to 1959. During the four and a half to five months of training, Dr. Holinka reportedly spent 5 to 6 hours per day working in a laboratory. The alleged asbestos exposures occurring during training included asbestos Bunsen burner pads and components from incubators; however, no exposures were alleged to have occurred during the first two months (basic training) and during the last two months of his training. Bunsen-burners were used approximately 2-hours per week and the pads were replaced "once they became brittle or somewhat dusty". Dr. Holinka alleges that he used an incubator for bacterial cultures and that the incubator may have contained components manufactured from asbestos. After completing training, Dr. Holinka worked as a laboratory technician in biochemistry, hematology, and pathology at the 98 General Hospital in Germany. While stationed in Germany, Dr. Holinka alleged exposure from asbestos mittens and Bunsen burner pads. The pads were used on a daily basis and changed once per week due to observed wearing of the asbestos pad. Asbestos mittens were also used on a daily basis for short periods, only minutes in many cases. With the exception of wearing a mask while working with bacterial cultures, no respiratory protection was worn while training or working in the laboratory for the U.S. Army.

Academic Coursework and Research

As an undergraduate, Dr. Holinka majored in French literature and had a minor in physiology. He completed his degree requirements and graduated in two and a half years. During the course of his undergraduate education, Dr. Holinka alleges that he used asbestos mittens and Bunsen burner pads in approximately six laboratory courses in chemistry and physiology. The mittens were reportedly used several times a session; a session defined as meeting twice a week for 3 hours over twelve weeks.

From the fall of 1962 to late spring of 1963, Dr. Holinka attended Hunter College in New York. He reported using asbestos burner pads in one course that met for three hours once a week for four months. Dr. Holinka left after two semesters to attend medical school at McGill University in Montreal. He alleged no use of asbestos products while at McGill, which lasted only two semesters.

From August of 1964 to August of 1968, Dr. Holinka was enrolled as a graduate student in physiology at UC Berkeley. He alleges exposure to asbestos mittens and burner pads while

performing research for his dissertation but the frequency and duration were not provided. His research was focused on rat brain endocrinology. After completing this degree, Dr. Holinka continued further studies in comparative literature, a program where he had no opportunities for exposure to laboratory equipment.

From 1971 to 1974, Dr. Holinka was enrolled as a graduate student in biological sciences at SUNY Stony Brook. He reported using asbestos mittens and Bunsen burner pads while performing research in the anatomy department for his degree. His research focused on hormonal control of the maternal paramount in rats. Most of the experiments conducted for his research were behavioral experiments. Dr. Holinka alleges that burner pads were used on a daily basis and were replaced no more than once per month. The mittens were reportedly used once every few days and were replaced approximately every four months.

Part Time and Temporary Laboratory Employment

After leaving military service, Dr. Holinka worked for 3-months (40hrs/week) as a laboratory technician at Booth Memorial Hospital. His work included clinical chemistry and analysis of human material serum and urine. Alleged asbestos exposure occurred as a result of his handling of asbestos mittens and Bunsen burner pads. Burner pads were used on a daily basis and were reportedly replaced every few days due to wearing of the pad. Mittens were used on a daily basis to handle hot glassware

While attending UC Berkeley as an undergraduate, Dr. Holinka worked part-time (12-20 hrs/week) in a research laboratory from the spring of 1960 to summer of 1962. His responsibilities were generally limited to analysis of California soils. He alleges use of asbestos burner pads and mittens. The burner pads were changed once ever two to three weeks. The mittens were reportedly used several times a week to handle hot glassware. Dr. Holinka described using mittens to swirl a flask while heating solutions and to remove glassware from a hot drying oven.

In the winter of 1964, Dr. Holinka left medical school and started working full time (40 hrs/week) in the same laboratory he had worked in as an undergraduate. He remained a full time employee of the laboratory until August of 1964. This time period of employment is not reflected in the social security records that were provided. During this period of time, Dr. Holinka allegedly used asbestos mittens and burner pads. From the end of 1961 until the beginning of 1971, Dr. Holinka did not work for a private employer as evidenced by his social security records.

Dr. Holinka was employed part-time (18 hrs/week) at the Columbia University Presbyterian Medical Center from 1971 to 1974 while pursuing his doctorate in biological sciences at SUNY Stony Brook. While working in this laboratory, Dr. Holinka's primary responsibility was the analysis of human serum plasma. Dr. Holinka alleges that he used asbestos mittens once every two weeks and burner pads on a daily basis.

Post-Doctoral and Faculty Research

From 1974 to 1977, Dr. Holinka worked as a post-doctoral fellow at the University of Southern California. This was primarily a research position; however, five to ten percent of his time was devoted to teaching. His research included animal work and biochemical

analysis and he would wear a surgical mask while performing experiments. Dr. Holinka reported using asbestos burner pads and mittens during this period of time.

After completing his post-doctoral fellowship, Dr. Holinka worked as a research instructor and later as a research professor at Mount Sinai from August 1977 to July 1989. He was involved in animal research and biochemical research in women's health care. Asbestos burner pads and mittens were used on a daily basis. Burner pads were replaced once every two months.

After 1989, Dr. Holinka began employment in the pharmaceutical industry with a number of firms and then became an independent consultant to the industry. He did not have any known asbestos exposures during this period of his career.

Summary of Opportunities for Exposure

Dr. Holinka provided limited information on the exact amount of time that he handled the Bunsen burner pads and the mittens that he claimed to use routinely. From his depositions, Dr. Holinka's first alleged asbestos exposure occurred while training and working as a laboratory technician in the U.S. Army from 1956 to 1959. From 1959 to 1963, Dr. Holinka had limited intermittent asbestos exposure while working with asbestos mittens and burner pads in academic and part-time employment laboratories. No alleged asbestos exposure occurred during the two semesters that Dr. Holinka was enrolled in medical school. From 1964 to 1966, Dr. Holinka again had limited intermittent exposure to asbestos mittens and burner pads. No alleged asbestos exposures occurred while he was pursuing a degree in comparative literature from 1966 to 1968 or while taking literature courses from 1968 to 1971. From 1971 to 1989, Dr. Holinka had limited exposure to burner pads and asbestos mittens. No alleged asbestos exposures have occurred since 1989.

In all, based on the very limited information that Dr. Holinka provided, I have estimated that his daily handling of the Bunsen burner pads would have been only about 60 minutes per day, three days per week for 20 years (taking into consideration the fact that he had several years where he did not have any potential exposure, did not work in the laboratory full time for much of this time, and had periods of time where he did not handle the pads on a daily basis). Likewise, his use of mittens for handling hot glassware would also be no more than 60 minutes per day, but the usage was likely only two days per week for 20 years (again considering that he did not use the mittens on a daily basis, did not work full time in the laboratory, and had time periods where he did not perform any lab work).

From his depositions, it was not possible to identify any other sources of asbestos exposure from his academic or professional careers.

Exposure Assessment

Asbestos Mittens

There have been studies of the use of asbestos containing gloves and mittens similar to the products used by Dr. Holinka. One study that has been frequently cited is the 1981 study by Samimi on asbestos exposure from wearing asbestos gloves. As part of the study, Samimi reported concentrations of airborne fibers emitted in five actual workplace laboratories that would have been similar to the laboratory environment in which Dr. Holinka worked. The

results ranged from 0.07 f/cc to 2.93 f/cc (mean = 0.83 f/cc; 7 samples). Samimi noted that the gradual soiling of gloves reduces the extent of fiber emissions although prolonged use could result in damage or deterioration. In discussing the range of measurements obtained from the five workplace laboratories, the study authors assert that differences in room size and arrangements, room ventilation system, and amount of moisture on the gloves are factors that influence exposure of workers. Gloves were composed of asbestos cloth containing 80-85% asbestos and 15-20% rayon and were treated with an acrylate-base compound to make them "lint-free." The fiber type was not specified in the study.

One of the major limitations of the study by Samimi is the use of the Phase Contrast Microscopy (PCM) analytical methodology. The PCM method is the most common method for the measurement of asbestos fibers in air and continues to be used extensively today. However, a significant limitation is that it does not distinguish asbestos fibers from other fibers. Although optical counting methods using membrane filters had been employed previously, the standard PCM methodology was not established by the National Institute for Occupational Safety and Health (NIOSH) until 1977. It was superseded by NIOSH Method 7400 around 1984. The approach to determining the level of fibers is defined by fiber counting rules. The fiber counting criteria include the counting of only fibers equal to or longer than 5 microns and the counting of all particles *as asbestos* (emphasis added) that have a length-to-width ratio (aspect ratio) of 3:1 or greater (NIOSH 1994). As stated in the documentation of the method, other airborne fibers (that is, non-asbestos fibers) may interfere, since all particles meeting the counting criteria are counted (NIOSH 1994). Thus the presence of gypsum, cement, silica, mineral wool, fiberglass, cellulose and other natural and man-made particles can, and often are, counted and treated as if they were asbestos. Consequently, an analysis by PCM indicating elevated fiber counts does not necessarily indicate the presence of asbestos nor the true magnitude of the exposure. At best, PCM merely provides an index of exposure to particles present in a given size range and shape, not necessarily that those fibers are asbestos (Baron 2001).

Another major limitation is that no background samples were reported to have been collected in the laboratories where Samimi conducted his measurements prior to the sampling to assess ambient fiber levels. Without controlling for ambient fibers, such as clothing, human hair, fibrous glass, or other fibrous matter, the asbestos exposure levels obtained in the Samimi study most likely overstate the true concentration.

Samimi also conducted testing in isolation chambers, which resulted in concentrations ranging from 0.95 to 11.74 f/cc. The range of results from the isolation chambers, which are substantially greater than the modern OSHA PEL, were most likely due to poor quality assurance methods that were in place at the time of the study and potentially poor housekeeping practices between simulations (Cherrie 2005). I am aware of some modern simulations that were carried out in isolation chambers that had high ambient fiber backgrounds, specifically a study by Dr. William Longo, of gaskets in which the background concentration in one of the simulations exceeded the OSHA PEL (Longo 2002). This type of poor quality control could explain the elevated results that were found during the sampling in the isolation chambers.

In a 2005 study on asbestos exposure from wearing asbestos mittens, Cherrie collected measurements during three separate glass manufacturing tasks. Chrysotile asbestos mittens

made in the 1970s were used. This study simulated three test conditions that would involve aggressive handling of materials while wearing mittens, methods that would be more aggressive than would be typical of use in a laboratory. In this study, the authors provided detailed information as to how they controlled for non-asbestos fibers through a process of cleaning and vacuuming the chambers after each simulation.

The tasks were performed both with no ventilation and with high ventilation within a 45 cubic meter enclosure. Laboratories typically have high ventilation rates due to the presence of fume hoods and the handling of chemical and biological agents. Ventilation requirements in the current American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE 2004) for laboratories require 1.0 cubic foot of air volume for every square foot of laboratory space. This requirement is greater than for nearly all other occupancies, except of automobile garages. The presence of fume hoods places additional demand for replacement air that can result in up to 10 air changes per hour in laboratory spaces (DiBerardinis 1993), again a level that is higher than most other occupancies.

The reported mean personal airborne fiber concentration from 33 samples ranged from 0.03 f/cc to 0.48 f/cc. The lowest mean fiber concentrations were obtained when high localized ventilation was used, and the highest mean concentrations were obtained when no ventilation was used. Both new and aged mittens were utilized, and the differences between mean airborne fiber concentrations for aged gloves and new mittens were not statistically significant. Based on observations made during the tests, obvious releases of airborne dust occurred when the mittens were abraded on sharp metal edges. Each simulation was carried out over a 30-minute period, and each task investigated was continuously repeated during that period. Cherrie found levels of fiber release from gloves, but ultimately concluded that the levels are not indicative of increased risk. As with the Samimi study, the authors used the PCM method for analysis, which could overstate the levels of fiber in the air. In addition, these results were not weighted for an 8-hour work day.

I have been provided with a workplace simulation conducted by Dr. Longo, provided to me in another matter. While Dr. Longo has used methods that are not consistent with accepted IH practice in other workplace simulations that he has undertaken, such as using Tyndall lighting and indirect preparation of samples for transmission electron microscopy analysis, my review of this simulation found that his methods and interpretation were in general conformance with good IH practice. In this simulation, gloves composed of 75% chrysotile asbestos were used. This simulation involved the repeated handling of bricks, which would result in aggressive and abrasive methods that would not be typical for laboratory use. In the simulation, 12 bricks were moved while wearing the gloves and the activity was repeated once each hour for four hours. The task based concentration results from this simulation were reported to be 0.02 f/cc.

It is my opinion that the data from the Cherrie and Longo studies are the best representation of Dr. Holinka's likely exposure from the use of the mittens. Based on the Cherrie study and the Longo simulation, it is my opinion that a reasonable maximum task based exposure would be 0.02 f/cc and a reasonable maximum estimate of the TWA exposure for Dr. Holinka, based on the frequency of glove use (which was infrequent, intermittent and irregular), would be 0.001 f/cc. With a daily average in this range for fifteen years, the reasonable maximum contribution to Dr. Holinka's lifetime cumulative exposure from his

use of asbestos mittens would be no more than 0.02 fiber-years/cc. The fiber type would have been chrysotile asbestos.

Bunsen Burner Pad

There have been no studies of the potential for the release of asbestos from the use of Bunsen burner pads in the peer-reviewed scientific literature. Bunsen burner pads were composed of iron wire mesh gauze, which came in various sizes and had a thin, small diameter, white circular center that contained asbestos. The white coloration of the center material is an indication that it is chrysotile containing as discussed previously. This thin layer of asbestos was designed to protect the bottom of the glassware from the high heat of a Bunsen burner flame.

While the fiber type and percentage of asbestos content in the pad is not known, it was most likely chrysotile due to the preponderance of chrysotile use in the United States. In addition, the center of the pad would not be friable, that is, not easily crushed or pulverized to powder by hand pressure, and would not release fibers under normal handling due to the binding of the asbestos fibers within a solid matrix. Using the pads as intended would not release fibers readily when used in normal laboratory heating procedures. It would require aggressive actions like sanding, grinding or abrading the center to release fibers, an activity that was not done by Dr. Holinka.

I have had personal first hand experience in the use of these Bunsen burner pads both in my academic experience and as a result of laboratory health and safety audits that I have conducted throughout my career. From my personal experience in a laboratory, the actual time spent handling the pads is minimal, only minutes per day. The pad is placed on a ring stand upon which a flask, beaker, or other type of glassware would be placed. The Bunsen burner would be placed underneath. During the heating of the glassware, there is no opportunity to come into contact with the pad. If running multiple tests, the pad would be left on the ring stand. Once the tests were completed, the pad would typically be stored once it had cooled and could be safely handled.

Dr. Holinka repeatedly stated that he believed that ambient asbestos fibers were likely generated as the heat from the Bunsen burners caused the fibers in the burner pads to become brittle. He could not recall the temperature of the Bunsen burner, nor could he recall the fuel source for the burners. Based on his descriptions, it is my opinion that the flaking and damage of the pads was due to thermal degradation of the asbestos due to the application of high heat.

A Bunsen burner is a gas burner commonly used in laboratories, most typically using natural gas as a fuel source, which can produce a flame capable of reaching temperatures of 1,500° Celsius (°C) or 2,732° Fahrenheit (°F) or higher (Bunsen burner 1998; Flinn 2007). All forms of asbestos are subject to melting and thermal degradation at temperatures beginning at 600 °C. Chrysotile will decompose to forsterite, a member of the olivine mineral family and a material that is not asbestos, starting around 500 °C with conversion to well-crystallized forsterite at temperatures from 800 °C (Jeyaratnam 1994) to 850 °C (ATSDR 2001; Vitra 2002). The amphibole forms also degrade with exposure to high temperatures. Amosite will degrade in to spinel, hematite and cristobalite starting at 600 °C. Crocidolite

will degrade into acmite, hematite and cristobalite starting at 800 °C. Heating for as little as 30 minutes at temperatures of 900 °C will result in the breakdown of all asbestos (Jeyaratnam 1994). These degradation reactions are not reversible.

As described by Dr. Holinka, the pads would wear out and he would replace them, sometimes as often as once every few days. Being subjected to frequent high heating with the Bunsen burner, at temperatures that were at or above 900 °C, the degradation reported by Dr. Holinka was reasonably the result of the breakdown of the asbestos fibers in the pad to non-fibrous, non-asbestos forms that are not linked to the development of mesothelioma.

Dr. Holinka's opportunity for exposure to asbestos from the use of the burner pads was irregular, intermittent and very limited. On average, he would have used these pads no more than a few hours per week, in many cases reasonably only minutes per day, with many periods of non-use. While the pads may have worn out frequently and needed to be replaced, this does not mean that he was exposed to asbestos fibers. He did not grind or pulverize the pads and did not take actions that would have readily released the asbestos fibers from the binder material.

The burner pads were not subject to aggressive handling other than the heating. If the use of woven gloves, picking up bricks, as simulated by Dr. Longo, could not create levels of asbestos greater than 0.02 f/cc, there is no possibility that the brief handling of intact burner pads could create levels higher than 0.02 f/cc. Any particulate that would be released from used pads that had been heated repeatedly would not be asbestos, having been degraded by the heat to non-asbestos forms. It is my opinion that, with a task based exposure that would be no greater than 0.02 f/cc for no more than 60 minutes per day for about three days a week, a reasonable maximum estimate of the TWA exposure for Dr. Holinka, based on the frequency of pad use (which was infrequent, intermittent and irregular), would be 0.0015 f/cc as a daily average. The reasonable maximum contribution to Dr. Holinka's lifetime cumulative exposure as a result of his use of the Bunsen burner pads would be no more than 0.03 fiber-years/cc.

Exposure Summary

The total cumulative exposure that Dr. Holinka would have had from the use and handling of the two primary laboratory products identified in his depositions has been reasonably estimated to be no more than 0.05 fiber-years/cc and with a high degree of certainty that the true exposure was in fact lower than this estimate. This level is still less than the cumulative exposure that a person living 70 years in an urban environment would receive from the presence of asbestos that is naturally present in the air. This level is also nearly two orders of magnitude less than the lifetime occupational exposure that a person working in an environment with TWA concentrations at the current OSHA PEL would receive.

Opinion

It is my overall opinion, within a reasonable degree of professional and scientific certainty, that Dr. Holinka's pleural mesothelioma was not the direct result of exposure to any asbestos containing products identified by Dr. Holinka. It is reasonable that the products allegedly used by Dr. Holinka were only chrysotile containing, a material that is not known

to have the potency for inducing mesothelioma, particularly at the exposure levels that would have been present in the laboratory environments in which he worked. Even at higher levels, the association of chrysotile to mesothelioma is weak. The 8-hour TWA concentrations and the lifetime cumulative exposures he had would be insignificant and irrelevant to the development of mesothelioma.

Further, it is my opinion that he had little or no exposure to asbestos from the brittle Bunsen burner pads due the asbestos undergoing thermal degradation because of the routine heating to elevated temperatures of greater than 900 °C. Asbestos present in the pad would be converted to other non-asbestos mineral forms, such as forsterite, that are not associated with a risk of mesothelioma development.

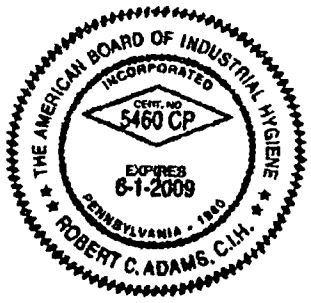
It is my opinion that there was no substantial asbestos exposure in Dr. Holinka's academic and professional career to explain the development of his pleural mesothelioma.

If you have any questions please do not hesitate to call me at (609) 243-9848.

Respectfully yours;

Robert C. Adams

Robert C. Adams, MS, CIH, CSP
Senior Manager
ENVIRON International Corporation



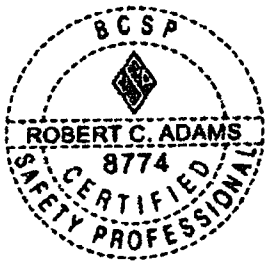


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Exhibit B

ENVIRON

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Subject: **In Re: New York City Asbestos Litigation**
Christian Holinka
Index No. 114120-06

Dear Counsel for the Defense:

Thank you for referring the above matter to me for epidemiological assessment. It is my understanding that the Plaintiff, Dr. Christian Holinka, claims his pleural mesothelioma resulted from exposure to asbestos from the handling of laboratory equipment and materials, including asbestos mittens and asbestos burner pads, allegedly obtained from Defendants ManorCare Health Services, Inc. (alleged to be a successor in interest to Central Scientific Company, a division of Cenco, Inc.) ("ManorCare"), Fisher Scientific International Inc. ("Fisher"), Baxter Healthcare Corporation (alleged to be a successor in

interest to American Hospital Supply Corp. and American Scientific Products) ("Baxter"), VWR International, Inc. ("VWR") and Univar USA Inc. ("Univar"), hereinafter collectively referred to as the "Lab Supply Defendants."

Following is my expert report in this matter, detailing my credentials, the methods used and materials relied upon, a critical review and synthesis of the relevant epidemiological literature, application of the scientific assessment to the facts apparent in this case, as well as my scientific opinions.

INTRODUCTION

Qualifications

I am by training and experience an epidemiologist. I was trained at the Master's level at the School of Public Health, University of Massachusetts and at the doctoral level at the School of Public Health, University of North Carolina. For ten years I served first as Assistant Professor and then as Associate Professor of Epidemiology in the Department of Biostatistics and Epidemiology of the School of Public Health and Health Sciences, University of Massachusetts. In 1991, I founded Applied Epidemiology, Inc., Amherst, Massachusetts, which in November 2003 merged with ENVIRON International Corporation, where I am a Principal, and serve as Director of Epidemiology. I have special interest and experience in matters pertaining to workplace exposures to various materials and chemicals including asbestos.

I have extensive experience in, for example, designing, conducting and publishing primary epidemiological research; critical review and synthesis of published epidemiological literature; the graduate training of epidemiologists, including classroom teaching, advising and chairing of Master's and Doctoral Committees; and serving in advisory, review and editorial capacities at the local, national and international level. I serve as Adjunct Professor in the Department of Epidemiology, University of North Carolina at Chapel Hill. I serve as Adjunct Associate Professor at Georgetown University, where I have team-taught on several occasions "Epidemiological Applications to Population Health" in the School of Nursing and Health Sciences. I am also Adjunct Associate Professor and member of the Dean's Advisory Board of the School of Public Health and Health Sciences, University of Massachusetts, where I teach on occasion. I am a Fellow of the American College of Epidemiology, and serve as Vice Chair of the College's Finance Committee. I also serve on the editorial board or as a reviewer of several scientific journals.

I have taught methods for critical review and synthesis of epidemiological studies as part of the core curriculum for Masters and Doctoral candidates in Public Health, and have used these methods to evaluate associations between various exposures and health outcomes. I have applied the same critical review approach to my evaluation of the epidemiological literature on occupational asbestos exposure and risk of mesothelioma, which is summarized below.

A copy of my current Curriculum Vitae which provides additional details as well as a list of my publications is attached.

Overview of approach

I have evaluated the peer-reviewed, published epidemiological literature on the relationship between exposure to various types of asbestos and the risk of mesothelioma, including the consideration of its very long latency (time between exposure and disease occurrence), to determine whether exposure to asbestos-containing laboratory supplies was likely to have caused Dr. Holinka's pleural mesothelioma. I have employed standard and widely accepted methods for critically and comprehensively reviewing and synthesizing the published, peer-reviewed epidemiological literature, and formulated my scientific opinions and conclusions based on this analysis. In addition to the relevant peer-reviewed, published epidemiological literature, I draw upon my education, training and professional experience, as well as on my analysis of materials provided to me by counsel for Lab Supply Defendants, including but not limited to interrogatory responses and deposition transcripts, to formulate my professional opinions and the conclusions offered. A bibliography of all materials relied upon is included below. I expect to review and comment upon additional scientific publications, documents, testimony, expert reports, exhibits and discovery related to the topics of this report as they become available.

METHODS

The epidemiological approach

Epidemiology is the field of public health that includes the study of incidence, prevalence, and distribution of disease in human populations, and factors that may be related to disease occurrence. It is a science that employs standard methods to identify and interpret statistical correlations, called "associations," between disease occurrence and other factors. Epidemiological research results are central to the determination of the role of specific risk factors in the general causation of disease in humans, and are broadly relied upon by epidemiologists and other professions as a basis for decision-making, including development of policy and judgments regarding specific causation. The validity of the epidemiological evidence and the validity of its interpretation determine the reasonableness of causal judgments that rely upon such evidence.

Epidemiological research addresses whether a disease is associated with specific exposures in groups of people or populations. Exposures measured on an individual basis provide the strongest evidence of an association, if it exists, between the exposure of interest and disease. Analytic techniques also can control for, or reduce possible effects of other exposures (i.e., confounding factors or confounders) that may be related to the exposure of interest and the disease under investigation. Epidemiologists are generally concerned with the impact that bias, due to systematic error, might have on study results. Systematic error in the methodology or due to missing or inaccurate information can render results invalid or even misleading, possibly related to how the groups being compared have been defined. Factors determining the quality of epidemiological studies

include the ability to avoid biases such as selective participation of certain subsets of individuals (selection bias), systematic errors in responses or measured data (information or misclassification bias), and identification of other risk factors for the same outcome that are correlated with the risk factor of interest (i.e., confounding bias).

Though the potential for bias exists in all studies, some study approaches and research settings are more prone to bias than others. The degree to which these challenges are addressed and overcome in an epidemiological study partly determines the strength and validity of the study results. Interpretation of the epidemiological literature also considers the role of chance in the results. Statisticians and epidemiologists evaluate the probability that an observed result is due to chance by applying tests of statistical significance. If the results are not statistically significant, chance cannot reasonably be ruled out as an explanation for the reported association (i.e., typically accepting a 5% error rate).

Dose-response assessment, or the evaluation of the relationship between estimated or actual dose of exposure and the disease risk, is a key tool of epidemiology. Ideally, the dose estimate is derived taking into account a reasonable period of disease latency. Latency is usually described as the time elapsed between the first known or possible exposure to the agent of interest (often indicated by date first employed in a particular job where exposure is likely) and the diagnosis of the disease of interest. This is also known as maximum latency, and is influenced by the ability to validly identify first exposure as well as the ability to detect the disease soon after it occurs. For many cancers that produce solid tumors, the latency is usually 20 or more years, and can exceed 50 years (as with mesotheliomas). If exposure is assessed without regard to disease latency, some or all of the exposure evaluation may be irrelevant to the occurrence of the disease.

Assessments of dose-response and latency are frequently used to inform causal judgment and policy formulation. The greatest impediment to such analyses is that many epidemiological studies fail to, or cannot, accurately characterize first possible exposure and the specific level or concentration to which each individual in the study was exposed over time: in fact, most studies that consider exposure rely upon surrogate measures such as employment history.

In addition to assessing the methodological quality of individual studies, weight of evidence syntheses consider the overall breadth and quality of the literature available. Just as results of individual studies might reflect systematic biases, a body of literature might be biased because of its focus on a chosen or preferred research hypothesis or study approach, or as a result of selection for publication. Such "publication bias" occurs when authors preferentially submit, and journals preferentially accept, studies demonstrating positive findings, even if such studies may be positive due to methodological weakness (e.g., small study size) or errors (e.g., exposure misclassification). Null study findings, even if based on well-designed and conducted studies, are considered "less interesting" and are less likely to be published (Hennekens and Buring 1987). The overall bias will be exaggerated if null findings contradict positive findings and/or if a weak study appears to support (or replicate) previously published or "accepted" associations.

Use of epidemiology for judgments of disease causation

Where a balanced and complete literature is available, it is possible to characterize that literature as reasonably supporting or failing to support a causal interpretation. If the weight of evidence favors a judgment of general causation, i.e., that a risk factor under certain conditions is capable of causing the disease in adequately exposed persons, epidemiological research can further help determine whether specific exposure attributes and other risk factors are more or less likely to contribute to risk among exposed persons. These risk factors may include but are not limited to the following:

- Dose or exposure concentration
- Type or form of exposure (chemical composition or physical structure)
- Timing of exposure (year first exposed, duration of exposure, etc.)
- Host susceptibility (genetic polymorphisms)
- Host attributes (age, sex, ethnicity, etc.)
- Co-exposures (viruses, smoking, etc.)
- Co-morbidity

Epidemiology generally cannot directly ascertain specific causation, i.e., whether a specific exposure played any role in the disease of specific individuals. Some cases of disease are idiopathic, and arise regardless of an individual's exposure history. However, understanding at a group level those factors that are associated with increased risk of disease may improve our ability to identify high or higher risk groups so that reductions in exposure can be targeted and implemented to prevent future disease. Based on these methods, we may also improve our ability to determine which subgroups are at increased risk due to their exposures, and by extension (and for non-scientific purposes such as decision-making and litigation) determine that the relationships observed at the group level may reasonably be applied to individuals who are in the study group or an adequately similar population. The determination of causation, however, remains a judgment, and cannot be proven. Although direct experimental evidence may offer stronger support for causal inference, experiments on health effects of toxic materials on humans are unethical. Toxicological or other laboratory data collected from animal models are useful for understanding the mechanisms of effects and for developing hypotheses regarding human health effects, but are not directly applicable to humans. For these reasons, well-designed epidemiological studies have been identified as the preferred scientific support for regulatory agencies such as the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), which determine policies regarding possible adverse effects of exposures. For the same reasons, the determination that an association is causal in humans is most reasonably based on the availability and proper evaluation of good epidemiological data.

Epidemiological review of asbestos and malignant mesothelioma

For this report, PubMed was used to identify key research reports and reviews published in the peer-reviewed medical/health literature that are relevant to the topics of this case. Provided by the US National Library of Medicine, PubMed is a powerful standard research tool available free over the internet, which searches the medical literature published since about 1966. The keywords "asbestos," "crocidolite," "amosite,"

"amphibole," "chrysotile," "pleural," "mesothelioma," "occupation," and "laboratory" were entered in various forms and combinations to identify a universe of potentially relevant articles available in the English language. Abstracts of the articles identified were screened for relevance, and articles meeting at least minimal criteria (i.e., article reports results of an epidemiological study, standard research methods were utilized, study population was exposed to asbestos, analyses included consideration of occupational asbestos exposure level, duration of exposure, type of industry, type of asbestos present, or other risk factors that help explain the risk of mesothelioma) were more thoroughly evaluated. Occupational studies of the primary and secondary asbestos industries were included in the review and are summarized below, as these studies examine populations of workers with documented and often heavy asbestos exposures and provide a considerable basis for understanding and evaluating potential risks among groups with lower exposures, including presumed and background levels of exposure.

EPIDEMIOLOGY OF ASBESTOS AND MALIGNANT MESOTHELIOMAS

Overview

Malignant mesotheliomas are cancers that arise most often in the pleura, the thin layer of tissue that surrounds the lungs and lines the chest cavity, or the peritoneum, the thin layer of tissue that surrounds the abdominal cavity and lines the abdominal organs (Blot and Fraumeni 1996). The risk of all types of mesothelioma combined is about 11 per million population per year in the US, with about 3,000 new cases diagnosed each year (Antman, Hassan et al. 2005). The incidence of cases not thought to be due to occupational exposure has been estimated at 1-2 cases per million person years (Bertazzi 2005).

Tumor registry data show an increase in the number of all types of malignant mesotheliomas, combined, beginning in the 1950s (Blot and Fraumeni 1996). Incidence increased sharply beginning in the 1970s and continued to rise through the mid-1990s (Price and Ware 2004; Bertazzi 2005). The increase has occurred almost exclusively among white men, as the rate for women in the U.S. has been stable since the 1970s at about 5 per million per year. Rates for non-white men have also remained relatively stable over time (Bertazzi 2005).

Risk factors for mesotheliomas include exposure to amphibole asbestos, erionite (a fibrous mineral from the zeolite group) and thorium dioxide (Thorotrast) – a radioactive contrast medium used in X-ray diagnostics until the 1950s in the U.S. "Amphibole" refers to a group of five of the six fibrous minerals that are considered to be asbestos: crocidolite, amosite, anthophyllite, actinolite and tremolite. Amphiboles are characterized by thin, rod-like fibers. In contrast, the most widely used form of asbestos, chrysotile, is a serpentine mineral, with curly and pliable fibers. Simian Virus 40 (SV40) cells have been discovered in mesothelioma tumors, suggesting this virus might play some role in the development of mesothelioma (Carbone, Rizzo et al. 2000; Vilchez, Kozinetz et al. 2003). Neither smoking nor ionizing radiation is considered a risk factor for mesothelioma (Antman, Hassan et al. 2005).

Some mesotheliomas are idiopathic, meaning they are not due to exposure to any of the recognized risk factors for the disease. Estimates of the rate of idiopathic mesothelioma may be derived from population-based cohort studies, where between 10% and 50% of mesotheliomas detected occurred in the absence of identifiable asbestos exposure (Boffetta 1998; Antman, Hassan et al. 2005; Bertazzi 2005). A proportion of all occupationally asbestos-exposed individuals with mesothelioma are also believed to develop mesothelioma due to causes (including residential or childhood amphibole exposure) other than their occupational asbestos exposure. Some number of idiopathic cases is expected to occur even among individuals with a history of substantial amphibole asbestos exposure.

Asbestos exposure and risk of pleural mesothelioma

Among asbestos-exposed persons, the chief factors that influence the risk of pleural mesothelioma are asbestos fiber type, the time since first exposure to asbestos (latency), and the level or concentration of exposure (dose).

Exposure to amosite, crocidolite or other types of amphibole asbestos is the clearest risk factor for pleural mesothelioma. In contrast, epidemiological evidence suggests the association between chrysotile asbestos exposure and pleural mesothelioma risk, if any, is weak, and may be due to amphibole contamination.

The epidemiological literature indicates that average latency – i.e., the time between first adequately high exposure to amphibole asbestos and occurrence of mesotheliomas – is very long, and may be as much as 60 years. The duration of latency may depend on both dose and fiber type. Low exposures are generally associated with longer latencies. Occupational studies of amphibole exposure and mesotheliomas typically show a dose-response relationship, with low or no risks observed among those with the lowest exposures.

Low-level asbestos exposures are also ubiquitous in the general population (i.e., not occupationally exposed persons), and can arise from regional geological features and from fibers released into the environment from consumer and building products. Use of asbestos (predominantly chrysotile) -containing consumer and building products increased after World War II through the 1970s, at which time a ban went into effect on asbestos in some consumer products, and asbestos was voluntarily withdrawn from other products. Ambient asbestos exposures also are thought to have increased and then declined over this interval, parallel to the increase and decrease in use of asbestos-containing consumer goods, though ambient environmental levels remained far below those measured in some work places. The lack of an increase over time in mesothelioma risk among women and non-white men has been taken as evidence for a necessary threshold, greater than ambient environmental levels, for amphibole asbestos exposure to lead to increased risk of mesothelioma (Bertazzi 2005).

Occupational studies of primary asbestos exposure and mesothelioma

All primary asbestos industries have reported increased mortality from pleural mesothelioma or all types of mesothelioma combined. The primary asbestos industries

include **mining and milling** (de Klerk, Armstrong et al. 1989; Piolatto, Negri et al. 1990; Sluis-Cremer, Liddell et al. 1992; Liddell, McDonald et al. 1997), **asbestos cement manufacturing** (Thomas, Benjamin et al. 1982; Finkelstein 1984; Alies-Patin and Valleron 1985; Ohlson and Hogstedt 1985; Gardner, Winter et al. 1986; Hughes, Weill et al. 1987; Raffn, Lynge et al. 1989; Albin, Jakobsson et al. 1990; Neuberger and Kundi 1990), **textile manufacturing** (McDonald, Fry et al. 1983; McDonald, Fry et al. 1983; Peto, Doll et al. 1985; Dement, Brown et al. 1994), **insulating** (Seidman, Selikoff et al. 1986; Seidman and Selikoff 1990), **friction and insulation materials manufacturing** (McDonald, Fry et al. 1984; Enterline, Hartley et al. 1987; Newhouse and Sullivan 1989); and **filter assembly and manufacturing** (McDonald, Gibbs et al. 1978; Jones, Smith et al. 1980; Talcott, Thurber et al. 1989) . Studies conducted in the primary asbestos industries, where the broadest range of exposures can be found, offer the best opportunity to quantify mesothelioma risks.

Mesothelioma risk depends on asbestos fiber type

Chrysotile accounts for 95% of asbestos produced in the world (Harington 1991; Terracini 2006) and is the most commonly used asbestos fiber in the US. As noted above, however, risk of mesothelioma is much more clearly associated with amphibole asbestos exposure. Among the amphiboles, crocidolite is most strongly associated with mesothelioma risk. Intermediate risks are seen among workers exposed to amosite. Increased risk of mesothelioma from occupational exposure to chrysotile alone, however, is less apparent and not seen in many studies. The risk differential for mesothelioma at similar exposure levels of chrysotile: amosite: crocidolite has been estimated to be 1:100:500 (McDonald and McDonald 1996; Hodgson and Darnton 2000). In studies where mesothelioma risk appears to be increased among workers with high-level chrysotile exposures (e.g., asbestos miners), there is reasonable evidence that the risk derives from contamination by amphibole fibers, as comparable groups heavily exposed to pure chrysotile appear not to be at increased risk of mesothelioma (McDonald and McDonald 1996).

Crocidolite asbestos

In employees with predominantly or heavy crocidolite exposure, the relative risk of pleural mesothelioma compared to unexposed or less exposed employees is extremely high. For example, proportional mortality from mesothelioma was examined among 33 men employed during 1953 in a Massachusetts factory that manufactured cigarette filters containing crocidolite. Through 1988, a total of 28 deaths had occurred. Five were from pleural mesothelioma, with only 0.01 expected (RR=460; 95% CI 150, 1080) (Talcott, Thurber et al. 1989). Jones et al. investigated causes of death among 951 women who had used crocidolite to manufacture military and commercial grade gas mask filters during the 1940s. The authors reported 29/166 deaths were due to mesothelioma, with risk positively associated with duration of exposure. The most deaths in this group occurred during the middle 1970s, approximately 30 years after first exposure to crocidolite (Jones, Smith et al. 1980).

Initial reports based on a cohort study of 13,450 friction product workers who primarily manufactured brakes and friction materials between 1941 and 1979 identified 10 deaths from mesothelioma, 9 among those with known crocidolite exposure. There was no

excess among those exposed only to chrysotile, the predominant fiber type used at the factory (Newhouse, Berry et al. 1982; Berry and Newhouse 1983). An updated mortality study of the cohort reported a total of 13 mesothelioma deaths, 11 among those known to be exposed to crocidolite (Newhouse and Sullivan 1989).

Chrysotile asbestos

Studies of workers predominantly exposed to chrysotile fibers inconsistently demonstrated slightly elevated risks of mesothelioma mortality compared to non-exposed populations. McDonald et al. identified 4,137 male textile workers who were mainly exposed to chrysotile who were employed for more than one month between 1938 and 1958 at a plant in Pennsylvania. Fourteen out of 1,392 death certificates mentioned mesothelioma (McDonald, Fry et al. 1983). Although information about exposure to specific fiber types was not provided in the report, the authors attributed the mesothelioma cases to amphibole exposure (McDonald et al., 1983b). Dement et al. (1994) reported mortality for a similar South Carolina textile manufacturing plant cohort, including those employed for at least one month between 1940 and 1965, with vital status ascertained through 1990 (Dement, Brown et al. 1994). Two pleural mesothelioma deaths were reported among workers primarily employed in the spinning department, with latency of 37 and 34 years, and 25 and 32 years employment, respectively. A third pleural mesothelioma death was identified when mortality follow-up was extended through 2001. The most recent decedent also had been employed in the spinning department, with a latency interval of nearly 50 years (Hein, Stayner et al. 2007).

Among 1,058 Italian chrysotile asbestos miners employed since 1946, two deaths due to pleural mesothelioma were identified among 427 total deaths – one in a worker with 20 to 30 years since first exposure and the other in a worker with more than 30 years since first exposure (Piolatto, Negri et al. 1990). One pleural mesothelioma was reported in a study of 2,167 chrysotile asbestos cement workers (Gardner, Winter et al. 1986) and no mesothelioma deaths were reported by McDonald et al. (1984) among 3,641 US friction product workers where chrysotile was the predominant fiber used (McDonald, Fry, et al. 1984).

It remains unclear whether the occasional mesothelioma cases seen among heavily chrysotile-exposed workers are due to high-intensity chrysotile fiber exposure, resulted from contamination by amphibole or unrecognized amphibole exposure from other sources, are of idiopathic origin, or reflect some combination of these (Elmes 1994; Britton 2002).

Mixed fiber types

Prior to World War II, insulation workers in New York and New Jersey were primarily exposed to chrysotile fibers. Subsequently, however, amosite was added to some of the insulation. In a cohort study of New York and New Jersey insulators, those working for long periods after the war were most likely exposed to amosite, an amphibole, and were at substantially increased risk of mesothelioma. No increase in risk was observed among those only exposed to chrysotile prior to the war, or those exposed to both chrysotile and amosite after the war but for less than 20 years (Selikoff, Hammond et al. 1979). This latter observation suggests that some exposure threshold (concentration and duration)

may be required before risk is meaningfully elevated; however, evidence of such a threshold has not been consistently observed.

In a 2006 publication, Yarborough reviewed studies of occupational cohorts, including some of the studies cited above, with exposures to various asbestos types and to mixtures of asbestos fiber types (Yarborough 2006). By combining cohorts, a total of 32,000 employees were identified who had exposures thought to be relatively pure chrysotile. There were only seven mesothelioma cases among these employees, and Yarborough found reason to question the accuracy and adequacy of the exposure evaluation and/or diagnosis for each of them.

Lung fiber burden analyses generally indicate exposures to mixtures of fiber types, even if work histories are unable to document mixed exposures (Yarborough 2006). For example, McDonald et al., (1997) examined dried lung specimens from chrysotile miners and millers in Quebec who died of mesothelioma. Of the 27 mesothelioma cases, 21 dried lung specimens were available. The investigators found tremolite and chrysotile fibers in 14 specimens from cases who worked in the Thetford mines, and chrysotile, tremolite, crocidolite and amosite in specimens obtained from the remaining 7 cases who worked in the mines in Asbestos (McDonald, Case et al. 1997). This investigation indicates that mesotheliomas that were attributed to chrysotile exposure may be due to the amphiboles contaminating the chrysotile at specific mining sites.

Although they offer suggestive evidence, lung fiber burden studies can be criticized on the grounds that fibers found in the lung at autopsy have an unknown relationship to fiber exposures that actually led to mesothelioma induction. Epidemiological evidence suggests that at least 30 years, and perhaps as much as 50-60 years, typically elapses between initial exposure and the onset of mesothelioma (see discussion of latency, below). Furthermore, amphiboles are expected to be found many years after exposure has ceased due to their persistence in lung tissue, whereas chrysotile fibers are relatively rapidly broken down and eliminated (Churg and DePaoli 1988; Britton 2002; Bernstein and Hoskins 2006). It is the relative durability and persistence of the amphiboles that is believed to contribute to their carcinogenic potency.

Exposure (Dose)

The relationship between mesothelioma and either intensity or duration of exposure, or a cumulative (intensity and duration combined) exposure estimate, is complex. Because exposure measurements are frequently unavailable in epidemiological studies, duration of employment or other surrogates are often used as indicators of "dose." Occupational studies that adequately account for disease latency and fiber type generally report a positive association between mesothelioma risk and either duration of employment or quantitative dose estimates. For example, of 8,009 deaths investigated among workers in the asbestos mines in Quebec, 22 of 25 mesothelioma cases were among men employed 20 or more years in the Thetford mines, and 5 additional cases were among men employed for at least 30 years in the Asbestos mines (McDonald et al., 1997). In a later publication, it was reported that the rate of mesothelioma increased with increasing fiber-years of exposure (Liddell, McDonald et al. 1997). Among 6,506 crocidolite miners/millers in Western Australia, the relative risk was 10.5 (95% CI 3.12-35.1) for

pleural mesothelioma among those with more than 6 months of exposure compared to those with shorter duration of exposure (de Klerk, Armstrong et al. 1989).

Among asbestos cement workers, Albin et al (1990) reported an increasing risk of pleural mesothelioma with increasing cumulative exposure compared to unexposed workers: RR=1.9 for workers with an average dose of 3.1 f/ml-years; RR=21.2 for an average dose of 25.6 f/ml-years; and RR=22.8 for average dose of 88.2 f/ml-years (Albin, Jakobsson et al. 1990). Similarly, Finkelstein (1984) reported an increasing trend in mesothelioma (all types) mortality with increasing dose among asbestos cement workers. The mean cumulative exposure for eleven pleural mesothelioma cases was 42 f/ml-years, and 161 f/ml-years for eight peritoneal mesothelioma cases (Finkelstein 1984). Among amosite-exposed factory workers, the lower the dose (time worked), the longer the time required for development of disease (Seidman, Selikoff et al. 1986).

Fiber type remains an important consideration when assessing the risk of mesothelioma associated with exposure concentration or duration. Among a cohort of South African miners, workers were exposed to crocidolite, amosite, or mixtures of both fibers (Sluis-Cremer, Liddell et al. 1992). Of the 30 cases of mesothelioma identified, 20 occurred among those with crocidolite exposure (none with less than 12 months exposure), 4 among those exposed to amosite for more than 3 months, and 6 among those with mixed exposures for more than 3 months. Cumulative exposure averaged 15.2 fibers/ml/year for the amosite sub-cohort (cases and non-cases), compared to 9.6 fibers/ml/year for the crocidolite sub-cohort (cases and non-cases).

Time since initial exposure (latency)

Time since initial exposure to amphibole asbestos is a strong predictor of incidence of mesothelioma. In the PMR study of cigarette filter manufacturers described above, the median interval from first exposure to crocidolite asbestos until mesothelioma death was 34 years (range, 26 to 37) (Talcott, Thurber et al. 1989). Any mesothelioma deaths that occurred after 1988 would only increase the average estimated latency, as cases with longer latency would not yet have been detected as of 1988, when the study was completed. In general, when mortality is used instead of disease incidence or diagnosis, the overall duration of latency will be somewhat exaggerated. Because the average survival time after mesothelioma diagnosis is short (about 1 year) (Stewart, Edwards et al. 2004; Antman, Hassan et al. 2005), the effect of using death rather than incidence or diagnosis to calculate estimated latency for mesothelioma will be limited.

In Australia, the incidence of malignant mesothelioma lagged 20 to 30 years behind trends of amphibole (primarily crocidolite) exposure. Incidence of mesothelioma for 1964 to 1968 among those 35 years or older at diagnosis was less than 1.0 case per million person-years, increasing to 15.5 cases per million person-years in 1979-1980. Among men aged 65 to 74 in 1979-1980, mesothelioma occurred in 69.7 cases per million person-years (Musk, Dolin et al. 1989).

Among 10,918 chrysotile miners and millers in Quebec first employed in 1904 and followed until 1992, a total of 38 mesotheliomas were reported – 33 in miners and millers and 5 in factory workers. Of these, 21 occurred in workers from the Thetford mines and

in Asbestos mines and mills – both sites among the earliest operations in Quebec (Liddell 1997). Average latency was 47 years, with a range of 21 to 60 years. PMRs increased with year of death: no mesothelioma deaths occurred before 1950, but subsequent mesothelioma death rates were 0.18% (1950-1974), 0.68% (1975-1984) and 1.10% (1985-1992). Rates of mesothelioma were significantly greater at the oldest Thetford mines (35.3/100,000 subject years) than at the Asbestos mine and mills (13.2/100,000) (McDonald, Case et al. 1997), possibly because of the longer follow-up time.

A mortality study of 4,137 textile manufacturing workers employed more than one month between 1938 and 1974 and exposed to chrysotile, amosite and crocidolite identified 14 mesothelioma deaths between 1960 and 1975. With the exception of one subject, deaths occurred 25 to 53 years from first employment (McDonald, Fry et al. 1983).

Initial studies of the insulation workers in New York and New Jersey followed through 1976 reported no mesotheliomas among those with less than 20 years exposure; 7 mesotheliomas occurred among those with 20 to 34 years, and 31 mesothelioma cases in New York/New Jersey insulators occurred after 35 years of exposure. Among a larger cohort of US and Canadian insulation workers followed through the same period, only 5 of 224 mesotheliomas occurred between 5 and 19 years after onset of exposure; rates of pleural mesothelioma were 2.78 per 1,000 person-years for 35 to 39 years after onset and 5.47 per 1,000 person-years for peritoneal mesothelioma 45 or more years after exposure onset. Additional follow-up of the 17,800 asbestos insulation worker cohort found peak mortality from mesothelioma (5.1 per 1,000) occurred 45 years after first employment. Although excess deaths from all causes combined decreased over time, there was no apparent decrease in deaths due to pleural mesothelioma or for those with more than 40 years since onset of first exposure (Seidman and Selikoff 1990).

Based on these studies, it is apparent that the greatest risk of mesothelioma occurs on average 40-50 years after first substantial exposure to amphibole asbestos occurs. Lower exposure concentrations would be expected to require even longer latency.

Although amphibole asbestos exposure is the most likely risk factor for mesothelioma, information on prior asbestos exposures of any kind may be sought specifically when mesothelioma is diagnosed. Information on specific occupational and other sources of asbestos exposure may be less actively sought for diseases or causes of death that have not been strongly associated with a specific exposure. Actively searching for any source of asbestos exposure when a diagnosis of mesothelioma is generated, or differentially seeking such information from cases and controls, would erroneously create or exaggerate observed associations between mesothelioma and asbestos exposure. These forms of bias may most impact studies of occupational groups less clearly or consistently exposed to amphibole asbestos.

Exposures among laboratory workers

Occupational health effects have been investigated among chemists and other laboratory professionals, including those working in academic or research settings as well as the chemical, biological, biomedical, and pharmaceutical industries. Although laboratory work settings are expected to include a wide variety of specific exposures that differ by

industry or research area (e.g., chemicals, biological materials, radiation), the basic equipment and supplies used in the laboratories are likely to be similar. A brief review of the literature on laboratory workers indicates that several studies have identified possible work-related health risks , including some types of cancer (Olin 1978; Olin and Ahlbom 1980; Cordier, Mousel et al. 1995; Edling, Friis et al. 1995; Brown, Paulson et al. 1996; Gustavsson, Reuterwall et al. 1999; Kauppinen, Pukkala et al. 2003; Dolan, Youk et al. 2004). The extent to which observed cancers were associated with any specific laboratory exposure is unclear; however, these studies have not identified increased risks for mesothelioma. This literature therefore does not identify laboratory workers as an occupational group at increased risk of mesothelioma.

SUMMARY OF RELEVANT FACTORS IN THIS CASE

Dr. Holinka was diagnosed with pleural mesothelioma in August of 2006. Based on an extensive critical evaluation of the epidemiological literature, it is clear that the main determinant of pleural mesothelioma risk is exposure to amphibole asbestos fibers, with risk dependent both on exposure intensity and latency. However, the literature also indicates that mesotheliomas occur in substantial proportion among persons with no known exposure to amphibole asbestos fibers. These cases may be idiopathic or the result of unrecognized exposures that might have occurred up to 40-60 years prior to diagnosis.

The following history can be reconstructed from Dr. Holinka's deposition testimony:

- Dr. Holinka first recalled asbestos exposure occurred in 1957, while training as a laboratory technician in the U.S. Army. During this two-month training period, Dr. Holinka worked in a laboratory for five to six hours per day and claims to have been exposed to asbestos through his use of Bunsen-burner pads (estimated at two hours per week) and incubators.

- From 1957 to 1959, Dr. Holinka worked in a laboratory while serving in the U.S. Army, and he alleged that he was exposed to asbestos through his daily use of asbestos mittens and burner pads.

- From 1959 to 1966 and from 1971 to 1974, Dr. Holinka reported using asbestos mittens and burner pads during intermittent periods of full-time and part-time employment, as well as during laboratory coursework conducted as part of his undergraduate and graduate studies.

- In 1974, Dr. Holinka started his post-doctoral fellowship and continued working in an academic laboratory while employed as an instructor and subsequently (1977-1989) as an assistant professor at Mount Sinai School of Medicine. Dr Holinka testified that he used asbestos mittens and burner pads throughout his fellowship and during most of his time working as an instructor and assistant professor. As an instructor and assistant professor, Dr. Holinka reported using asbestos mittens once per day and changing worn out burner pads about once every two months.

The alleged sources of asbestos exposure described by Dr. Holinka in his deposition testimony were mittens (allegedly made of or containing asbestos) and Bunsen-burner pads with a center disk of asbestos, both used in general laboratory settings. Dr. Holinka testified that the asbestos from the burner pads would become brittle and flake off after a period of use and exposure to heat. Once wear was apparent, the existing pad would be replaced with a new one. Dr. Holinka reported that the frequency with which he changed the burner pads ranged from "every few days" to "once every two months" depending upon the use of the pads and how quickly they became brittle. Dr. Holinka reported no direct knowledge of the manufacturers or suppliers of any of the laboratory materials and equipment alleged to contain asbestos.

Assuming that the mittens and burner pads did contain asbestos, the asbestos type contained in these products would most likely have been chrysotile. If, as alleged by Dr. Holinka, the wearing of the Bunsen burner pads occurred as a result of subjecting the pads to heat, it is likely that the brittle material that composed the flakes was not asbestos, but rather a non-asbestos mineral such as fosterite formed by thermal conversion of asbestos. Normal use of mittens and Bunsen burner pads would be expected to generate only very small quantities of respirable chrysotile fibers. Exposure to chrysotile – even in substantial concentrations – has not been associated with increased risk of mesothelioma; therefore, intermittent exposure to low concentrations of chrysotile fibers would have had no bearing on Dr. Holinka's mesothelioma.

CONCLUSIONS

Based on my review, analysis and synthesis of the published epidemiological, occupational health and case-specific information available to me, I conclude to a reasonable degree of epidemiological certainty that Dr. Holinka's pleural mesothelioma did not result from using asbestos mittens and burner pads. Due to the lack of identifiable exposure to amphibole asbestos, Dr. Holinka's mesothelioma is likely of idiopathic origin. However, as in any mesothelioma case, it is also possible that Dr. Holinka's mesothelioma was induced by an undocumented exposure to amphibole fibers, possibly occurring 50 or more years ago.

Please do not hesitate to contact me if you have questions or require further information.

Sincerely yours,

Kenneth A. Mundt, Ph.D.
Principal and Director of Epidemiology
ENVIRON International Corporation

Case-specific materials relied upon

Document Title	Description
Moline Report	Dr. Jacqueline Moline expert report
Answers to Interrog	Letter of Application (complaint) and Plaintiff's Answers to Interrogatories
Social Security Records	Social Security Records
Holinka Depo I	Deposition under oral examination of Christian Holinka
Holinka Depo II	Deposition under oral examination of Christian Holinka (Volume II)
Holinka Depo III Plaintiff's Expert Report	Deposition under oral examination of Christian Holinka (Volume III) Dr. James Strauchen, MD expert report Expert for the Plaintiff Pathologist
Holinka CV	Curriculum Vitae of Christian Holinka
Medical Records - Dr. Meyers	Medical Records from Dr. Robert Meyers
Medical Records – NY Presbyterian Hospital	Medical Records from New York Presbyterian Hospital
Medical Records – Dr. Taub	Medical Records from Dr. Robert Taub at the Herbert Irving Cancer Center
Medical Records – Radiology	Medical Records from Columbia Presbyterian Eastside Radiology
SSN Records	Social Security Records
Defendant's Expert Report	Robert C. Adams, MS expert report Expert for the Defendant Industrial Hygienist

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